volume depletion. The mean weight loss in the NT was 1.22 kg, and in the HPT was 1.36 kg. None of the patients had orthostatic hypotension, and their response to isometric exercise was normal. The NT had a normal response to the Valsalva maneuver (mean VR, 1.57; controls VR, 1.81) and a normal BBV (BBV, 160 msec; control BBV, 245 msec). In the HPT, the response to the Valsalva was significantly impaired (VR, 1.18; $P < 0.005$), and there was an equally marked reduction in the BBV (BBV, 56.6 msec; $P < 0.005$). There was ECG evidence of ischemic heart disease in one and left ventricular hypertrophy in two of the HPT. The VR showed a negative correlation with increasing MAP ($r = -0.57$). Acute volume depletion made no difference to the performance of either group, although three of the HPT were clinically volume-overloaded before dialysis. Thus, we conclude that the absence of orthostatic hypotension and the normal response to isometric execise imply a largely intact autonomic nervous system. In spite of this, the VR and BBV were abnormal in the HPT, and it is assumed that unless there is a parasympathetic defect, other factors, possibly of cardiac origin, must be responsible. Both the Valsalva maneuver and BBV alone are inadequate tests of autonomic function.

A study in patients with recurrent renal stones associated with hypercalciuria and assessment of the response to treatment with sodium cellulose phosphate. *M. L. Zaltsman, A. M. Meyers, L. Laidley, J. Pettifor, and F. J. Milne. Renal Unit, Department of Medicine, University of the Witwatersrand and Johannesburg General Hospital and Metabolic Research Unit, Department of Paediatrics, Baragwanath Hospital, South Africa.* Twenty-one percent of admissions to the urological service at the Johannesburg General are for the treatment of primary calcium calculi. Pak et al categorized recurrent renal stone-formers into absorptive, resorptive, and renal hypercalciurias. Other workers have not found a high incidence of hypercalciuria in these patients. Therefore, this study was undertaken to further clarify this point, to categorize pathogenetic mechanisms, and to accurately gauge the effect of sodium cellulose phosphate in the treatment of this common problem. Three groups were investigated: *1)* normal controls, *2)* idiopathic hypercalciurics, *3)* patients with either hyperparathyroidism or sarcoidosis. All patients in group 2 had a history of *recurrent* urinary calculi, and all patients were randomly referred and had 24-hr urinary calcium excretions of > 300 mg/24 hr. Subjects were placed on a diet containing 600 to 800 mg of calcium/day for the duration of the study. Tests were carried out before and six weeks after commencement of treatment, and in order to assess the efficacy of cellulose phosphate, all subjects were followed more than one year. The following investigations were carried out: *1)* calcium 47 absorption, *2)* total calcium and phosphorus, *3)* ionized calcium, *4)* 8-hr urine test of Edwards, *5)* 6-hr one-gram calcium load of Pak, *6)* serum hydroxycholecalciferol levels, *7)* 24-hr urinary calcium and cAMP levels. The absorption tests showed clear-cut differences between the groups (viz., group 1, 2.9%; group 2, 3.68%; and group 3, 4.86% of the administered dose/liter of plasma). Urine isotope levels also were different in the groups (group 1, 0.54%; group 2, 1.09%; group 3, 0.9% of administered dose). Mean ionized calciums were: group 1, 2.21 ± 0.11; group 2, 2.13 ± 0.14 ($P < 0.05$), and group 3, 2.91 ($P < 0.001$). Serum proteins were comparable in all groups. Serum hydroxycholecalciferol levels were normal in all groups. A clear-cut difference between the three groups was also seen in the urine calcium: creatinine ratios, returning towards normal after treatment in group 2. These, plus cyclic AMP results and the 8-hr urine test results will be presented and discussed. We conclude that these tests indicate various pathogenetic mechanisms and allow clinical categorization of patients with hypercalciuria. No cases of normocalciuric or renal hypercalciuric stone-formers were found. Two patients with "normocalcaemic" hyperparathyroidism were unearthed and successfully treated. Treatment with sodium cellulose phosphate improved biochemical abnormalities, and the rate of new stone formation was decreased markedly in all but one patient.

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Mild glomerulitis with pneumopathy and *Pneumococcus* bacteriemia. *H. Beaufils, P. Solal-Celigny, D. Uzzan, M. Lucsko, J. Guédon. Service de Néphrologie, Service de Pneumologie, Suresnes, France.* A 50-yr-old man with *Pneumococcus* bacteriemia was admitted to the hospital for a pneumopathy. During the course of the disease a macroscopic hematuria developed lasting three days, and persisted microscopically during three weeks. Urinalysis revealed a proteinuria (1 g/24 hr) for five days, without hypertension or renal failure. A renal biopsy, performed 22 days after the first renal symptom, showed a moderate mesangial hypertrophy without hypercellularity and extramembranous deposits ("humps") with deposition of C3. Serum C3, C4, and C3PA were normal on two occasions, and cryoprecipitate was not detected. The titer of antistreptolysin 0 was unexpectedly high, 1,200 U to 800 U. The presence of circulating immune complexes was suggested by the inhibition of complement-dependent lymphocyte rosette formation by the serum of the patient. This observation is an example of a glomerular disease occuring during the course of a visceral infection with bacteriemia.

Progressive dialysis myoclonic encephalopathy: Responsibility of tap water. *F. Cartier, P. Allain, D. Chevet, M. Garré, J. P. Hervé, and S. Pecker. Clinique des Maladies Infectieuses. Unité de Néphrologie. Hôpital Pontchaillou, Rennes, France.* A progressive encephalopathy, mainly characterized by myoclonus speech disorders and occasionally seizures, occurred in 14 chronic hemodialysis patients, 12 of them dialyzed in the same dialysis center. With the aim of determining the cause of this syndrome, a comparative study was made between patients with encephalopathy, patients without encephalopathy who were dialyzed in the same center or at home, and some control cases without chronic nephropathy. Brain grey matter aluminium values in patients with encephalopathy were 5.5 μg/g of wet tissue, as compared with 1.5 μg/g in controls ($P < 0.001$). White matter manganese values were also increased : 0.66 μg/g as compared with 0.41 in controls ($P < 0.01$). Copper, zinc, and iron contents were not different. Blood dialysate, and tap water aluminium levels were significantly higher in this center than those of home dialysis patients. Dialysate and end dialysis blood aluminium levels were linearly correlated ($P <$

0.001), but cumulative intake of aluminium containing salts was not higher in patients with encephalopathy than in other dialyzed patients, and it was not correlated with blood aluminium levels. These data suggest that this syndrome may be due to metals, particularly aluminium, contained in tap water used for dialysate preparation. Aluminium and manganese should be measured in tap water repeatedly, so that the water can be adequately treated if necessary.

Hereditary C_2 deficiency in patient with lupus erythematosus. *M. Dantant, M. Godin, C. Rivat, D. Gilbert, M. Fontaine, D. Dubois, B. Cavelier and J. P. Fillastre. Unité I.N.S.E.R.M. U78 and Service de Néphrologie, Bois Guillaume, France.* A new case of C_2 deficiency in a French family is reported. HLA typing, polymorphism of properdine factor B (Bf), and study of total complement and fractions showed that gene C_2d for C_2 deficiency segregates both with the HLA-A10=B27/Bf^s haplotype and with the HLA-A9=B7/Bf^s haplotype. C_2 deficiency was associated with an SLE syndrome in a girl; her brother, with the same C_2 deficiency, was an apparently healthy boy. Gene C_2d was known to be associated with haplotypes HLA-A10-B18, -A9-B18, A11-B18, and A10-Bw40. All these haplotypes contained either the B18 or the A10 allele. However, association between C_2d gene and other haplotypes which did not contain the A10 or the B18 alleles were also found: -A2-B12, -A3-B5, A9-B5, -Aw30-B13, -A1-B8, -A2-B5. In this case, two additional haplotypes inherited together with the C_2d gene were reported. Bf and C_2d are found very close to each other; C_2 deficiency was thus always found associated with the Bf^s allotype.

Hypoglycemic activity of the main metabolite of glibenclamide: Influence of renal insufficiency. *J. Fabre, L. Balant, L. Loutan, and H. Samimi, Policlinique universitaire de médecine, Genève, Switzerland.* The administration of 5 mg of ^{14}C-glibenclamide (GLI) in seven subjects with renal insufficiency showed that a complete hepatic transformation of GLI into its metabolites limited the accumulation of the original molecule. On the contrary, the urinary excretion of the metabolites was reduced (normal, 28% of the dose in 24 hr; GFR of 32 ml/min, 18%; GFR of 7 ml/min, 2.3%). Therefore, it was important to know if the metabolites have a hypoglycemic activity. Groups of six rats with ligatured ureters received i.p. 1 mg/kg of GLI, or of its main metabolite, 4-trans-hydroxyglibenclamide (OH-GLI), or saline. Each rat was paired with a control animal that had a sham operation. The serum glucose was measured during 4 hr after injection (glucose oxydase micromethod). In the controls, GLI caused a significant decline of the glycemia ($P < 0.05$), but less marked that with OH-GLI ($P < 0.001$). Dose-effect curves showed that the relation of the ED_{30} (dose necessary to reduce the glycemia by 30%) for GLI and OH-GLI is 6.5. Therefore, the metabolite has a strong activity, not demonstrated until now, 50 to 100 times greater than tolbutamide under the conditions of this experiment. In rats with ligatured ureters, OH-GLI causes a greater hypoglycemic response than in the controls ($P < 0.001$). *Conclusions.* Renal insufficiency, in man, slowed the excretion of OH-GLI. This metabolite, in the rat, had a marked hypoglycemic activity, which was increased when urinary excretion was suppressed. These data permit a better understanding of the pathogenesis of hypoglycemic manifestations in patients with renal insufficiency treated with glibenclamide.

Effort hemodynamic data in patients on chronic hemodialysis. *J. P. Fendler, G. Dongradi, J. C. Kahn, P. Rocha, and M. Ben Fahrat. Département de Néphrologie and Département de Cardiologie, Centre Hospitalier Intercommunal de Poissy, France.* In sixteen patients on chronic hemodialysis without patent heart disease or cardiac failure, hemodynamic data were determined at bed rest, in sitting position, and at the highest level performed on the ergometer bicycle (60 or 90 watts): mean right atrial pressure (RAP), mean pulmonary wedge pressure (PWP), a systolic arterial pressure (SAP), diastolic arterial pressure (DAP), heart rate (HR), cardiac output (CO), cardiac index (CI), systolic index (SI), and lactacidemia (LA). These parameters were correlated with SAP, DAP, hemoglobin (Hb), and arterioveinous fistula output (Δ OFAV). At the highest effort performed the values were: RAP, 5.6 $\pm$ 4.4 mm Hg; PWP, 17.2 $\pm$ 7.8 mm Hg; SAP, 208 $\pm$ 42 mm Hg; DAP, 98 $\pm$ 24 mm Hg; HR, 129 $\pm$ 19 beats/min; CI, 9.3 $\pm$ 2.0 l/mn-m^2; SI, 73 $\pm$ 15 ml/m^2; LA, 2.4 $\pm$ 1.1 mEq/liter. Maximum exertional performed CO is normal when compared to sedentary normals subjects; PWP is increased at bed rest but is no more increased for a maximum effort. It was not possible to demonstrate that either SAP, DAP, PWP, Δ OFAV, Hb, or lactacidemia might be the factors limitating the maximum exertional cardiac output.

Evidence for a cortico-renal substance inhibiting compensatory hypertrophy of the kidney. *Ch. Fontenaille, J. Lucas, F. Buzelin, and J. Guenel. Laboratoire de Pathologie Médicale et Néphrologie, U.E.R. de Médecine, Nantes, France.* Increasing number of cells accounts partly for 'compensatory'' growth of contralateral kidney after nephrectomy. We have tried to inhibit this hyperplasia by infusing into rats cytosol prepared from liver, whole kidney, cortex, or medulla. We have observed, by comparison with controls, a significant decrease of radioactive thymidine uptake by the left kidney in animals injected with cytosol of whole kidney or cortex but not in animals given medulla or liver cytosol. This experiment supports the assumption that compensatory hyperplasia would result from a lack of growth autoregulation normally mediated by renal cortex.

Effect of ultrafiltration on plasma levels of parathyroid hormone (PiPTH). *J. F. De Fremont, A. Fournier, J. Gueris, B. Coevoet, G. Lambrey, J. Quichaud, Amiens, France.* Lack of renal degradation of PTH contributes to the increase of PiPTH in uremic man. Polyacrylonitrile membrane (PAN) is permeable to PTH. Therefore PiPTH was measured in two patients (F,G) at monthly intervals by radioimmunoassay (normal range, 4--8 ng/ml) with plasma levels of calcium (Ca) and phosphate (PPO_4), before and after hemodialysis (HD) and then ultrafiltration (UF) on PAN. No difference was observed between the significant decrease of PiPTH during a HD with three liters of ultrafiltrate and a dialysate Ca of 7 mg/dl (−49%) and those observed during a UF of 18 liters with reinfusion fluids containing, respectively, 60 (55 $\pm$ 4%), 70 (53 $\pm$ 4%), 80 (52 $\pm$ 4%), and 90 (52%) mg of Ca. The ratio ultrafiltrate:plasma of PTH did not change between the beginning and the end of UF (0.37 $\pm$ 0.09 vs. 0.42 $\pm$ 0.10). Study of prefiltration levels showed the critical role of two parameters: the control of PPO_4 by phosphate binders and the Ca balance during a UF procedure. The plasma phosphate being constant, the negativity of this latter (−128 mg of Ca per procedure) led in one month to an increase of PiPTH from 93 to 146 ng/ml in patient F, whereas in patient G its positivity (+ 115 mg of Ca per procedure) led in one month to a fall of PiPTH from 140 to 92 ng/ml. It is stressed that Ca balance depends not only on Ca concentration in the reinfusion fluid but also on the weight loss, so that with the standard concentration of 7 mg/dl, this balance varies from −26 to +114 mg. The plasma phosphate being constant, PiPTH before UF was in the same range as before HD with PAN, when the mean Ca balance of the previous UF was 60 $\pm$ 5 mg. It is concluded that Ca balance during a UF procedure does not influence PiPTH after UF, a mirror of its clearance, but has a critical role on PiPTH before UF, which appears therefore as being mainly the mirror of PTH secretion rate. UF does not appear more effective than HD with PAN in clearing PTH or in controlling PTH hypersecretion in uremic patients.

Corticodependent hypercalcemia (HCa) in patients under regular dialysis treatment (RDT). *M. Fuss, J. L. Vanherweghem, P. Bergman, D. Verbeelen. Départment de Néphrologie, Hôpital Universitaire Brugmann, Brussels, Belgium.* Among 330 patients admitted to RDT, three developed persistent HCa (> 2 mg/dl) after one to six months of RDT. Original kidney diseases were glomerulonephritis, cortical necrosis, and lupus nephritis. All three patients were dialyzed with 7.5 mg calcium/dl dialysate and treated

with $Al(OH)_3$, but no vitamin D (VD) and no calcium supplements were given. Subtotal parathyroidectomy (PTX) was performed in two cases for vomiting and painful periarticular calcinosis, but PTX did not correct HCa. Parathyroid hormone plasma levels were normal in all three patients, even preoperatively in the two patients who underwent PTX. In one PTX patient, hypocalcemic tetany followed immediately prednisone (PDS) administration necessitated by renal transplantation. In the two other cases, HCa was corrected after a few days of moderate corticotherapy. Plasma 25(OH)VD was normal in both patients prior to PDS administration, but whole body retention of ^{47}Ca indicated high normal values for intestinal calcium absorption. Hyperparathyroidism and VD intoxication were thus excluded. High-calcium dialysate in HD patients with high normal calcium absorption could lead to HCa. Whether PDS acts by decreasing calcium absorption, or by reducing osteolysis, or by both mechanisms, is still unsolved.

Evidence of a hypovitaminosis PP in the source of chronic renal insufficiency treated by repeated hemodialysis. *A. Gross, D. Ponsin, B. Labidi, E. Prenat, J. P. Rivory, and J. P. Nicolas. Clinical and Experimental Nephrology, Faculté B de Médecine, Nancy, France.* Vitamin PP was measured systematically in the blood of 54 patients with chronic renal insufficiency treated by repeated hemodialysis. In all subjects, the concentration of nicotinic acid in the blood was very low compared to normal subjects, and this lowering was generally more marked after rather than before dialysis. The question arises whether this hypovitaminemia plays a part in the pathogenesis of some of the cutaneous, digestive, and nervous disturbances observed in these patients. A vitamin replacement therapy was instituted, the results of which are difficult to interpret and give rise to considerable discussion.

Clinical results obtained with a twice-weekly short duration dialysis strategy. *C. Jacobs, I. Reach, J. Rottembourg, and P. Degoulet. Service de Néphrologie, Groupe Hospitalier Pitié-Salpétrière, Paris, France.* An ultra short dialysis (USD) strategy, 2×5 hr per week, was used in 27 patients during a cumulative period of treatment of 333.5 months. 16 patients were treated with 1 m^2 surface area dialyzers with cuprophane membranes, 11 with dialyzers of > 1.40 m^2 surface area. The selection of patients and the choice of dialyzers were based on body weight, maximum daily urinary output, and creatinine clearance. In 12 patients, the results obtained by USD were compared with those recorded during a control period of six months' duration during which the patients were treated according to standard dialysis schedules. In six patients on USD with 1 m^2 dialyzers, the only significant modifications compared to the control period were a rise of predialysis plasma level of BUN and of postdialysis plasma levels of BUN and phosphorus. A decrease in motor nerve conduction velocity (MNCV) without clinical symptoms was noticed in one patient. In six patients treated on larger surface area dialyzers, only pre- and postdialysis values of BUN and creatinine were found to be higher than during the control period. Out of the group of 27 patients, nine (33.3%) had to be withdrawn from USD: two because of persistent hyperkalemia and/or hyperphosphatemia (associated with decrease of MNCV in one case), two because of pericarditis, one because of decrease in MNCV, one deliberately when affected by hepatitis. Elective indications for USD are hospital dialysis patients weighing 60 kg $\pm$ 10, with a daily urinary output of $\geq$ 800 ml, a residual creatinine clearance of $\geq$ 2 ml/mn. Close medical supervision is mandatory. USD allows a better rehabilitation of hospital dialysis patients, and an optimization of dialysis facilities.

Diagnosis of hemolysis and uremia in late pregnancy and puerperium: Acute tubular nephritis or thrombotic microangiopathy. *A. Kanfer, L. Morel-Maroger, A. Vandewalle, M. Godin, J.D. Sraer, O. Kourilsky, and G. Richet. Service de Néphrologie et Unité de l'INSERM, Hopital Tenon, Paris, France.* Acute hemolysis and thrombocytopenia occurred in eight of 24 patients admitted over a period of six years for acute renal failure (ARF) during late pregnancy or puerperium. Six patients had renal biopsy, showing in three of them acute tubular nephritis (ATN) and thrombotic microangiopathy (TMA) in the other three. In the three patients with ATN, pregnancy was complicated by preeclamptic toxemia or abruptio placentae, and ARF immediately preceded or followed delivery; two of the three patients had schistocytosis; the three patients made a complete recovery. In one of the three patients with TMA, ARF developed at the 22nd week of pregnancy complicated by toxemia; in the other two, pregnancy had been normal and the onset of ARF was delayed up to 2 and 12 weeks after delivery, respectively; two of the three patients had schistocytosis. The three patients received heparin therapy. Ultimately anuria was persistent in the two patients with TMS who had arterial-arteriolar lesions; a rapid and complete recovery occurred in the woman who had TMA limited to the glomerular tuft. This report indicates that the hemolytic-uremic syndrome of late pregnancy or puerperium may be related to ATN as well as to TMA. The clinical and hematological features of these two conditions may be indistinguishable. Renal biopsy may allow diagnosis and therefore prediction of outcome. Also it gives a basis for heparin therapy in TMA, which seems better indicated when arteries and arterioles are preserved.

Cancer and transplantation: An additional case report. *G. Lelievre, M. Ribet, B. Gosselin, Ph. Dequiedt, E. Lepoutre, A. Tacquet. Service de Médecine Générale et Néphrologie A, Hôpital Calmette, and Service de Chirurgie, Hôpital Calmette, and Laboratoire d'Anatomo-Pathologie, Hôpital Calmette, Lille, France.* We report the case of a 24-yr-old woman, who, because of a familial nephropathy, received a cadaver kidney transplant. Two months later she developed nodular lesions in the lungs. A surgical biopsy showed the metastatic nature of these lesions. In spite of interrupting immunosuppressive treatment, the patient died six weeks later. Autopsy study found a renal carcinoma in the transplanted kidney. The authors review the literature and discuss the pathophysiological consequences of this association between carcinoma and immunosuppression for transplantation.

Basophil sensitization in shunt nephritis. *A. Martini, M. Levy, M. Broyer, J.F. Hirsch, J. Benveniste. I.N.S.E.R.M. U.30 et 25, Service de Neurochirurgie, Hôpital Necker Enfants-Malades, Paris, France.* In rabbit acute serum sickness, the IgE-basophil system plays an important role in the deposition of immune complexes (IC) through the release of vasoactive amines from platelets and the consequent increase in vascular permeability. By contrast, little is known about the role of basophils in human glomerulonephritis. We have investigated the possibility of the sensitization of basophils in a case of glomerulonephritis secondary to an infected ventriculo-atrial shunt. The responsible antigen, i.e., coagulase-negative staphylococcus, has been clearly identified in this type of immune complex nephritis. The diagnosis of shunt nephritis was made on clinical, biological, and histological data in a 16-yr-old male. Coagulase-negative staphylococcus was cultured from blood and ventriculoatrial shunt. Twenty-five days after removal of the catheter, when blood cultures were sterile, we performed an *in vitro* degranulation test in the presence of either the bacteria cultured from patient's blood or a pool of coagulase-negative staphylococcus. A marked degranulation was observed in both tests. We have therefore demonstrated the presence of a basophil-dependent hypersensitivity towards an antigen previously present in the blood and, in all probability, implicated in the pathogenesis of glomerular damage. This finding substantiates the hypothesis that IgE-basophil-platelet system might in man contribute to IC deposition within the glomerular capillary walls.

Acute leukemia complicating renal transplantation. *C. Mendes da Costa-Richard, G. De Roy, Y. Thoua, E. Dupont, and J.P. Naets. Services de Médecine et d'Anatomie Pathologique, Hôpital Universitaire Brugmann, Brussels, Belgium.* A 39-yr-old male, with chronic interstitial nephritis, treated by kidney transplantation and immunosuppression (azathioprine and prednisolone) for 11 years, developed acute leukemia. After two months of dialysis, he received a first cadaver transplant in 1966. In 1969, after rejection of this graft and a short period of dialysis, he received a

second cadaver transplant. In 1973, chronic cytomegalovirus hepatitis supervened, but liver disease was well-tolerated and graft function was good, although with moderate leukopenia and thrombocytopenia. In March 1977, rupture of oesophagal varices, associated with major pancytopenia supervened, and azathioprine was discontinued. Blood and marrow smears showed numerous blasts, and the patient died with high fever and diffuse hemorrhages three months later. Several cases of leukemia have been described after renal transplantation and after immunosuppressive therapy alone. This unusual complication of transplantation could be due to allogenic stimulation, immunosuppression, or reactivation of oncogenic viruses.

Three cases of total parathyroidectomy and autotransplantation of parathyroid tissue in the forearm. *A. Meyrier, J.P. Jablonski, and G. Caillens. Service de Néphrologie, Hôpital Tenon, and Service de Chirurgie Infantile, Hôpital Trousseau, Paris, France.* In three cases of chronic renal failure the four hyperplastic parathyroid glands were removed, and 20 slivers of parathyroid tissue were transplanted intramuscularly in the forearm. Another parathyroid gland was preserved by deep freezing for a possible second transplant. Two patients were suffering from chronic renal failure of long duration. The third had been hemodialyzed for five years. All three had bone pain and pruritus. Severe osteoclastic resorption was conspicuous on X-rays and (in two cases) on the bone biopsy. The operation and the further clinical course were eventless. The main biological features were as follows:

Case no. 1

	Ca	P	Ph.alc. (N=< 85)	PTH (N=<1.2)
Preop.	7.5	5.0	180	11.5
D + 7	4.5	7.5	180	0.75
D + 90	7.5	5.0	50	1.2

Case no. 2

Ca	P	Ph. alc.	PTH
10.5	5.0	850	13
5.5	4.5	700	3
9.5	2.5	160	1.75

Case no. 3

Ca	P	Ph. alc.	PTH
9.5	5.5	550	20
5.5	3.8	500	1.5
9.0	3.5	60	2.4

The clinical and X-ray abnormalities regressed progressively. The control of hypocalcemia necessitated high amounts of oral calcium, 25-OH-D_3 and DHT. Comparative PTH radioimmunoassay in the brachial veins showed concentrations 1.5- to 20-fold on the autotransplanted side as compared with peripheral blood. The PTH secretion rate was stimulated by hypocalcemia and shut-off by hypercalcemia. This technique has the following advantages over 7/8 PTX: *1)* easy postoperative course; *2)* quantified amount of parathyroid tissue left in the body; *3)* possibility of 2nd operation under local anesthesia for adding or removing parathyroid fragments if necessary; *4)* PTH assay at its source.

The response of the renal kallikrein-kallidin system to uninephrectomy in rats. *A. Mimran, G. Baudin, D. Casellas, W.C. Wahbe. Department of Medicine, Montpellier, France.* The response of urinary kallikrein (BAEE esterase activity) to uninephrectomy and the changes of kallikrein to dietary sodium restriction in 1-week (group I) and 5-week (group II) uninephrectomized rats were investigated. In group I (N = 6), uninephrectomy was associated with a 40% decrease in urinary kallikrein excretion (U Kall) from 9.18 ± 0.2 to 6.38 ± 0.8 IU/24 hr ($P < 0.005$) seven days after nephrectomy. Kidney weight increased from 0.92 ± 0.03 to 1.13 ± 0.02 g ($P < 0.001$). Sodium restriction induced an increase in U Kall to 10.4 ± 1.5 ($P < 0.025$). In group II (N = 6), U Kall was 7.64 ± 0.8 (before nephrectomy), 4.93 ± 0.65 (1 week), and 6.7 ± 0.45 (5 weeks after nephrectomy). Kidney weight was 1.38 ± 0.02 g (1.10 ± 0.04 control, $P < 0.001$). After 7 days of sodium restriction, U Kall increased to 8.5 ± 0.8 ($P < 0.05$). In control normal rats, sodium restriction was associated with an increase in U Kall from 8.3 ± 1.2 to 15.2 ± 1.6 ($P < 0.001$). Uninephrectomy is associated with a progressive adaptation of urinary kallikrein excretion without any correlation with sodium regulation since UNa V did not change during the study in group I and II rats. However, uninephrectomy seems to blunt significantly (especially 5 weeks after nephrectomy) the response of renal kallikrein to sodium restriction. The role of a change in the response of the renin-angiotensin-aldosterone system to sodium restriction needs to be investigated.

Utilization of sodium acetate by patients on maintenance hemodialysis (MHD). *R. Oulès, B. Branger, M. Chevallet, C. Polito, G. Desch, B. Descomps, C. Mion. Service de Néphrologie, C.H.U. Nîmes et Service de Biochimie A, C.H.U. Montpellier, France.* In order to study the metabolic effects of the acetate (SA) load induced during hemodialysis (HD), plasma SA determinations were performed by gas chromatography in 34 uremics on MHD. Dialysis schedule was five hours, 3 times weekly on a Gambro Lundia 13.5=μ 1 m^2 dialyzer, with a constant Q_B at 190–450 ml/min, Q_D 500 ml/min, and dialysate SA at 38.5 mmoles/liter without dextrose. Three protocols were followed: *1)* Plasma SA was determined before and at the end of HD in 34 patients (2 or 3 times in 15 cases). *2)* Plasma SA kinetics were done during HD (5 cases) and after HD (3 cases). *3)* In 16 cases, plasma beta-OH-butyrate (BHB), acetoacetate (AA), lactate, and pyruvate were also determined before and at the end of HD. Results are as follows: *1)* Plasma SA level before HD was 0.6 ± 0.04 mmoles/liter, increasing significantly to 5.6 ± 0.68 (range 1.7 to 19.6 mmoles/liter) at the end of HD. There was no consistent influence of Q_B, but marked variations between dialysis sessions were found in the same patient. *2)* Plasma SA increased in all patients, achieving a steady state with a "plateau" in 5 cases and increasing continuously up to the end of HD in 2 cases. After HD was completed, a rapid decrease of plasma SA was observed with an estimated $t_{1/2}$ of 11 752 ± 150.9 μmoles/liter) as well as AA (59 ± 7.6 to 508 ± 90.8 μmoles/liter). There was no correlation between SA level and BHB and AA levels or BHB/AA at the end of HD, or Δ BHB, or Δ AA during HD. However, BHB and AA levels at the end of HD were correlated to the caloric intake before HD (r = − 0.61 and r = − 0.62, respectively). Lactate decreased during HD (1121 ± 103.7 to 796 ± 76.9 μmoles/liter) but not pyruvate (101 ± 14.7 to 98.9 ± 11.1 μmoles/liter). The results indicate a tendency to SA "accumulation" in some patients during HD, followed by disappearance from plasma after HD. This observation suggests a variable SA utilization rate among the group of patients. There is no apparent relation between the SA load and the increase of ketone bodies in plasma.

Mechanisms of renal compensatory hypertrophy. *F. Stephan, P. Reville, and F. de Laharpe. Hospices Civils de Strasbourg, Strasbourg, France.* The mechanisms of renal compensatory hypertrophy are still poorly understood. A renotropic substance may appear in the blood after loss of renal tissue. It is more likely that the blood level of an inhibiting substance is reduced. So hypertrophy and hyperplasia may occur until the amount of this substance is replenished by the growing tissue. According to this hypothesis, the kidney controls its own growth. The increased work load, essentially related to tubular sodium reabsorption, is probably not responsible for initiating kidney regeneration, but it may control the degree of renal compensatory growth. It may be modified by certain metabolic and hormonal disturbances: in the rat, renal compensatory hypertrophy is enhanced by administration of ACTH or by diabetes mellitus; it is reduced—but not abolished—by hypothyroidism.

Propranolol and pregnancy: Preliminary results of a prospective study. *Ph. Tcherdakoff, C. Kreft, E. Berrard. Hôpital Ambroise Paré, Boulogne, France.* Theoretically, beta-blockers may prove harmful during pregnancy: there may be increase of uterine contractions (risk of premature birth), fetal bradycardia, neo-natal hypoglycemia. A preceding retrospective study seemed to minimize these dangers. A prospective study with precise measurements of uterine contractions, fetal cardiac rhythm, neonatal glycemia, and APGAR index has been started in order to support the initial data. Preliminary results, obtained from a small number of patients, apparently show that the harmful effects of beta-blockers during pregnancy are less important than initially thought.

Repeat biopsies in membranous glomerulonephritis (MGN). *M. Zanetti, L.H. Noel, C. Barbanel and D. Droz. Clinique Néphrologique et Service de Thérapeutique Néphrologique, Hôpital Necker, Paris, France.* Fourteen patients with MGN had repeat biopsies. All but two had idiopathic MGN. All biopsies were studied by light microscopy; 18 were examined by immunofluorescent and 13 by electron microscopy or on thin sections. Three stages of lesions of the glomerular capillary wall were observed: *type I:* diffuse presence of subepithelial deposits (SED) without spikes; *type II:* presence of SED with diffuse spikes; *type III:* complex morphological changes of the capillary wall. On the first biopsy 7 patients were type I and 7 were type II. None had renal insufficiency. *Evolution: A) Complete pathological recovery* (three cases) was observed in two patients with type I and one with type II lesions, 1.5, 4, and 5 years, respectively, after the first biopsy. All patients had normal urinalysis and normal renal function at the time of the second biopsy. *B)* Three patients with type I had *unchanged lesions* 6 months to 2 years after first biopsy. Isolated proteinuria without renal failure persisted in two patients. One had clinical remission. *C) Change of type* (eight cases): One patient with type II lesions on the first biopsy had type I on a second biopsy performed 10 years later and subsequently had clinical remission. Two patients with type I on the first biopsy had type III when re-biopsied after an interval of 3 and 6 years. One developed renal insufficiency, the other had clinical remission. Five patients with type II on the first biopsy showed type III on the second biopsy. The intervals ranged from 6 months to 4 years after the first biopsy. One developed end-stage renal failure, three had clinical remission, and one had persistent nephrotic syndrome. In conclusion, the change from type I through type III does not always correspond to a progressive worsening of the clinical status of the patient. All morphological types may change and lead to complete pathological recovery.

Histopathological course in 55 cases of pregnancy nephropathy. *P. Zech, S. Colon, N. Blanc, M. Labeeuw, and J.P. Chalmet. Clinique de Néphrologie et des Maladies Métaboliques, Hôpital Edouard Herriot, Lyon, France.* Fifty-five patients with persistent abnormalities after pregnancy nephropathy (19 recurrent forms) were studied by 60 renal biopsies performed from less than 3 months (19 cases) to more than one year (9 cases) after the onset of the disease. Fifty single biopsies showed moderate glomerular proliferation or cellular swelling, segmental hyaline sclerosis microhyalinosis, and frequent mesangial hyperplasia. Subentothelial deposits, chiefly in the arterioles, appeared or increased with time. Hyperplasia of the juxtaglomerular apparatus was occasionally noted. Twenty-one immunofluorescent studies revealed arteriolar C3 deposits in 40% (before 3 months), 58% (between 3 and 6 months), and 56% (after 6 months) of the cases. Except in early biopsies, there was no glomerular immune deposition. Fibrin deposits decreased with time. Serial biopsies (5 cases) showed a decrease in the glomerular proliferation; mesangial hyperplasia and segmental hyaline sclerosis remained stable. Subendothelial deposits were frequently seen, mainly on the arterioles, and hyperplasia of the juxtaglomerular apparatus occurred in one case. Five early biopsies revealed vascular C3 deposits (2 cases) and glomerular fibrin deposits (4 cases). C3 deposits were found on all late biopsies. In conclusion, arteriolar lesions with C3 deposits were the main findings of prognostic value. They might be correlated with the subsequent rise of blood pressure, even without proteinuria.

Transient hypercalcemia associated with Reiter's syndrome in chronic dialysis patients. *J. Zingraff, B. Amor, T. Drüeke, N.K. Man, P. Jungers, and J. Crosnier. Clinique Néphrologique, Hôpital Necker, and I.N.S.E.R.M. U.25, Paris, France.* Reiter's syndrome, frequently associated with the presence of the intracellular parasite, *Chlamydia trachomatis A* in urethral specimens, has never been reported in dialysis patients. Four (about 10%) of our hemodialysis patients experienced unexplainable episodes of fever and acute arthralgias. Their association with conjunctivitis in all and with urethritis in three orientated us to the diagnosis of Reiter's syndrome. In all patients, intracellular inclusions highly suggestive of *Chlamydia* were demonstrated (in urethral specimens in three patients, in the conjunctiva in one). However, these inclusions were not cultured. Lymphoblastic transformation in the presence of a *Chlamydia* (ornithose-psittacose) antigen was negative in all, but the test was performed more than one year after the onset of symptoms. Screening for specific serum antibodies is in progress. Only one of the patients possessed HLA B27 phenotype. Three of these four patients had transient elevation of plasma calcium concentration during the acute phase of the syndrome (predialysis plasma calcium > 11.5 mg/100 ml). This elevation lasted for three to four weeks. The hypercalcemia might be related to increased bone resorption due to inflammatory periostitis. A comparable bone resorbing activity has recently been reported in hypercalcemic patients with rheumatoid arthritis. *To conclude,* the frequency of Reiter's syndrome may be underestimated in hemodialysis patients. One should evoke this diagnosis when unexplained fever, arthralgias and/or hypercalcemia complicate the course of long-term dialysis treatment. Further investigations on this syndrome should be completed by culture of the intracellular organism. A study of its epidemiologic distribution in dialysis patients would be of great interest.

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Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and cerebrovascular accidents. *V. Beroniade, and A. Farah. Department of Nephrology, Hôtel-Dieu de Montréal, Montreal, Canada.* The SIADH is frequently described as a complication of cerebrovascular accidents, but to our knowledge, there is no published study concerning its incidence and clinical implications. In a series of 20 patients, we found hyponatremia and hypoosmolality starting from the first to the fifth day in 15 patients. The severity of the hyponatremia is not always remarkable; in one case, however, the sodium plasma level decreased to 105 mEq/liter